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Prediction of adolescent smoking from family and social risk factors at 5 years, and maternal smoking in pregnancy and at 5 and 14 years

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ABSTRACT

Aims This study examines associations between maternal smoking and family, social or child risk factors when the child is aged 5 and adolescent smoking. The influence of mothers who smoke in pregnancy or continue to smoke at 14 years was also examined.

Design The Mater-University of Queensland Study of Pregnancy is a prospective cohort study.

Participants Participants included 8556 women enrolled between 1981 and 1984 at their first antenatal visit. Completed questionnaires were obtained for 7223 offspring, comprising the study birth cohort. Of the 7223 eligible children a total of 4541 had information on both maternal smoking when the child was aged 5 years and adolescent smoking at 14 years.

Measurements Measures included maternal smoking during pregnancy and when the child was aged 5 and 14 years, child smoking at 14 years, maternal alcohol use, child behaviour problems and social and demographic variables.

Findings Adolescent smoking was predicted by a risk score at 5 years involving maternal smoking and alcohol use, non-married status, having a partner who had ever been arrested, having four or more children in the household, and child aggression at 5 years. Continued maternal smoking from 5 to 14 years was associated strongly with adolescent smoking. There was also evidence that smoking in late pregnancy may exert an independent effect on adolescent smoking.

Conclusions The results suggest the possibility of a direct effect of prenatal smoking on adolescent smoking and highlight a set of environmental risk factors in the development of adolescent smoking. These risk factors may be used as early warning signs that intervention may be needed, and given the similarities with risk factors for other adverse childhood outcomes, the benefits of early intervention may extend beyond smoking to other problem behaviours. The possibility of being able to predict other disorders, because of these associations, also warrants further investigation.

Keywords Adolescent smoking, child risk factors, longitudinal, maternal smoking, prospective.

INTRODUCTION

It is during adolescence that most experimentation with cigarettes takes place (Chen & Kandel 1995; Chassin *et al.* 1996) and the earlier the age of smoking onset, the more severe the addiction to nicotine is likely to be (Taioli & Wynder 1991; Breslau & Peterson 1996). A number of studies have used risk factors to predict adverse outcomes and psychopathology in children (e.g. Sameroff *et al.* 1987; Fergusson, Woodward & Horwood 1998; Gutman, Sameroff & Cole 2003; Rutter 2005). While an examination of genetic transmission is beyond the scope of the current study, a wide range of risk factors was examined to assess the utility of developing a risk factor score as a means of predicting adolescent smoking.

Explanations of the mechanisms by which factors such as maternal smoking lead to adolescent smoking include genetic transmission of vulnerability and gene-environment interactions (Kahn *et al.* 2003; Rutter 2005; Taylor & Rogers 2005), damage to the central nervous system and long-term changes in the reward circuitry of the brain by exposure to nicotine in pregnancy (Niaura *et al.* 2001; Nestler & Malenka 2004), learned behaviour arising from observation of significant others (Avenevoli & Merikangas 2003; Slomkowski *et al.* 2005) and social explanations (Taylor 2003). These factors are not mutually exclusive and may indeed be part of the same causal sequence.

Retrospective reports of maternal smoking and offspring outcomes

Several studies have investigated the link between offspring smoking and retrospective reports of maternal smoking during pregnancy. Lieb *et al.* (2003) found that regular smoking by mothers during pregnancy, as well as by those who did not smoke during pregnancy, were associated with higher smoking rates in the children, although pregnancy smoking added an additional risk. Kandel *et al.* (1994) reported that daughters of mothers who smoked during pregnancy were approximately four times more likely to smoke in adolescence than either offspring of non-smokers or offspring of women who had not smoked during pregnancy. In a later study (Griesler, Kandel & Davies 1998), a relationship was found between maternal smoking during pregnancy and current smoking among adolescent daughters, potentially reflecting a direct biological effect. Finally, a longitudinal follow-up study of male twins aged 8-16 years found no association between maternal smoking and boys' smoking (Silberg *et al.* 2003).

Prospective reports of maternal smoking (assessed during pregnancy) and offspring outcomes

In a longitudinal cohort study, Fergusson *et al.* (1998) found that after adjustment for confounding variables, the only significant association relating to maternal smoking was that between maternal smoking and greater conduct disorder symptoms. In contrast, Kandel & Udry (1999) found that in 240 mothers and their 15—17-year-old daughters, self-reported prenatal maternal smoking, prenatal exposure to maternal testosterone and maternal smoking during daughters' adolescence directly affected adolescent smoking. In a 30-year prospective study, Buka *et al.* (2003) found that offspring (aged 17—39 years) whose mothers smoked one or more packs/day at some time during pregnancy had an increased risk of becoming dependent on nicotine. Finally, in a study of children followed from birth to 10 years, Cornelius *et al.* (2000) found that children exposed to more than half a pack of cigarettes per day during pregnancy had 5.5 times the risk of early experimentation with cigarettes.

Overall, there are important differences between studies in relation to the age at which offspring are assessed (e.g. early adolescence versus late adolescence to adulthood), the behaviour being assessed (e.g. initiation of smoking versus the development of dependence) and the extent to which confounding factors have been controlled. Based on prospective studies that have followed offspring into adolescence, it is possible that there is a direct biological link between maternal smoking during pregnancy and adolescent smoking. There is some evidence of a link between prenatal exposure to maternal testosterone and adolescent smoking, although the mechanism underlying this link remains unclear. Finally, in order to differentiate the effects of prenatal maternal smoking from the effects of potential confounding variables, factors such as maternal alcohol use, adverse parental and family factors and current maternal smoking should be accounted for. Current maternal smoking, for example, is important in role modelling and could be confounded with the effect of smoking during pregnancy. Only two of the studies, however, controlled for this variable and mixed findings were reported (see Kandel *et al.* 1999; Cornelius *et al.* 2000).

The current study involves the Mater-University of Queensland Study of Pregnancy (MUSP), a large study with detailed measurements of selected biological, psychological and social factors. The study is significant, in that we have assessed maternal smoking prospectively and taken measures from children at birth, 5 years and 14 years. A previous paper from this study (Conwell *et al.* 2003) had indicated that adolescent smoking was associated with a broad web of social, family, behavioural and educational disadvantage at 14 years. Our aims in this study were, first, to determine whether maternal smoking and family, social or child risk factors when the child is aged 5 are associated with adolescent smoking, and whether a risk score at 5 years would be useful in predicting adolescent smoking. Secondly, we examined whether the association with maternal smoking at 5 years was related to mothers who continued to smoke at 14 years, and finally, if maternal smoking in pregnancy was an independent predictor of adolescent smoking.

PARTICIPANTS AND METHODS

The Mater cohort

The MUSP cohort initially involved 8556 women enrolled between 1981 and 1984 at their first antenatal visit to the Mater Misericordiae Mothers' Hospital, Brisbane (see Keeping *et al.* 1989 for further details of the cohort). At the time of hospital discharge, completed questionnaires were obtained for 7223 children, comprising the study birth cohort. Mothers were interviewed at the time of the first pregnancy visit, shortly after delivery, at 6 months and at 5 and 14 years when the child was also assessed. Of the 7223 eligible children a total of 4541 had information on both maternal smoking when the child was aged 5 years and adolescent smoking at 14 years. The mean age at the 14-year follow-up was 13.9 ± 0.34 years with a range of 12.5—15.5 years.

Measures

Maternal smoking

This was assessed in early and late pregnancy by two questions: (1) In the last week, how often did you smoke? Responses consisted of every day, every few days, once. and not at all. (2) In the last week, how many cigarettes did you usually smoke each day? Responses were categorized as nil, one to nine, 10—19, ≥20/day. Similar questions were asked at each follow-up visit.

Adolescent smoking
This was also assessed with two questions: In the last week, how often did you smoke (every day, every few days, once, not at all)? How many cigarettes did you smoke last week? Responses consisted of nil, one to nine, 10—19, 20—29, 30—49 and 50+0. They were then categorized as nil, one to nine per week and 10/week.

Other variables
Frequency of maternal alcohol intake was measured at 5 years with the following question: How often do you drink alcohol? Responses consisted of daily, a few times a week, a few times a month, a few times a year, rarely and never. To assess quantity of consumption, respondents were asked, How much alcohol do you usually drink at those times? Responses consisted of seven or more glasses, five or six glasses, three or four glasses, one or two glasses, less than one glass, and never drink. Procedures for determining the daily average ounces of alcohol are described in an earlier publication (O'Callaghan *et al.* 2003). The four final alcohol consumption categories used in further analyses were nil, less than half a glass, half to less than one glass and more than one glass/day.

Maternal depression at 14 years was measured using the Delusions Symptoms States Inventory (DSSI; Bedford & Foulds 1978). Child behaviour problems were measured at the time of the 5-year assessment using 31 items from Achenbach & Edelbrock's (1981, 1983) Child Behaviour Check List (CBCL: Najman *et al.* 1997). The selected items were grouped according to the second order grouping of syndromes identified by Achenbach (1991) and similar to Achenbach, the extreme 10% were classified as having a behaviour problem. The Cronbach alphas were 0.76 for internalizing problems (INT), 0.84 for externalizing (aggressive) problems (EXT) and 0.75 for social, attentional, thought problems (SAT). Five of the 10 items in the SAT scale were measures of attention. The Peabody Picture Vocabulary Test—Revised (PPVT-R) was also administered, although to a smaller number of children. It is a measure of receptive language that correlates well with measures of verbal intellectual ability (Dunn 1981). As the focus of this paper was on predictors of adolescent smoking, it was examined as <85 (—1 SD) and ≥85.

Other measures at 5 years included marital status and gross family income, history of either parent being arrested, number of children in the household, quality of the marital relationship assessed by a modified Spanier's (1976) Dyadic Adjustment Scale and social network size of the mother. Maternal age and level of education were assessed at the first visit.

Statistical analysis

Categorical predictors of adolescent smoking at 5 years were examined using the x^2 test. Factors with $P < 0.1$ in the univariate analyses were then included simultaneously in a logistic regression model as predictors of adolescent smoking. Those factors

significant (<0.05) in this initial logistic model were then included simultaneously in the final model. Independent predictors from this final logistic model were summed without weighting to create an adolescent smoking prediction risk score. Strength of association was expressed as an odds ratio (OR) or adjusted (adj) OR and its 95% confidence interval (95% CI). The effect of adding to the model maternal cigarette smoking in early and late pregnancy and when the child was 14 years was also explored. In all models, smoking was entered as a dichotomous variable (no smoking/ smoking). A two-tailed P-value of <0.05 was used to assess statistical significance.

Test characteristics for different numbers of risk factors in the prediction risk score were examined using sensitivity (proportion of people with the target condition who have a positive test). specificity (proportion of people without the target condition who have a positive test) and positive predictive value (proportion of people with a positive test who have the target condition).

In order to account for those lost to follow-up, we first explored whether those remaining in the study significantly differed from those lost to follow-up. Measures that predicted loss to follow-up at age 14 included maternal age, education, marital status, income and smoking measured at baseline and birth weight. Assuming the data were missing at random (MAR; Hogan, Roy & Korkontzelou 2004), we applied the stochastic 'switching regression' method of van Buuren *et al.* (1999) to estimate imputed values for the missing items. Imputations were performed with the multivariate imputations by the chained equations (MICE) method recently implemented for the STATA program (Royston 2004). When we replicated our logistic regression models on the imputed data sets, results did not change substantively from the complete case analysis presented here.

RESULTS

Examination of study and non-study groups showed that mothers lost to follow-up were of lower educational level and age. were more likely to be unmarried and poorer, to have smoked more in early pregnancy and had children of lower birth weight (Table 1).

Table 2 shows the 10 family, maternal and child factors at 5 years related significantly to adolescent smoking. No relationship with adolescent smoking was present for maternal age, size of the maternal social network, quality of the marital relationship, child factors of PPVT-R <85 or presence of other child behaviour problems. All factors from Table 2 related significantly *(P* <0.1) to adolescent smoking were included simultaneously in a logistic regression model with any adolescent smoking at 14 years as the dependent variable. Seven independent predictors of adolescent smoking were found: maternal smoking (any), maternal drinking, non-married, low income, partner ever arrested, four or more children in the household and child aggression.

Table 1 Comparison of study and non-study groups

	Non study (n=2682)		*Study (n=4541)*		
	n	*%*	*n*	*%*	*P-value*
Maternal education					
Incomplete high	571	21.5	734	16.3	
Complete high	1693	63.7	2916	64.6	
Post high	392	14.8	864	19.1	<0.001
Maternal age (years)					
13-19	604	22.5	577	12.7	
20-34	1963	73.2	3760	82.8	
>34	115	4.3	204	4.5	<0.001
Marital status					
Married	1705	64.3	3681	81.7	
Single	381	14.4	355	7.9	
De facto	453	17.1	391	8.7	
Other	114	4.3	80	1.8	<0.001
Gross family income					
≤$10 400	1065	43.6	1243	28.9	
≥$10 400	1380	56.4	3061	71.7	<0.001
Maternal cigarettes early pregnancy					
Nil	1432	54.0	2963	65.8	
1-9	469	17.7	722	16.0	
10-19	431	16.3	503	11.2	
>20	319	12.0	313	7.0	<0.001
Child gender					
Male	1402	52.3	2356	52.0	
Female	1280	47.7	2185	48.1	0.7
Child birth weight (g)					
<1500	11	0.4	17	0.4	
1500-<2500	127	4.7	156	3.4	
≥2500	2544	94.9	4367	96.2	0.02

The seven factors statistically significant in this logistic model as independent predictors of adolescent smoking were then included in the final model (see Table 3 for the unadjusted and adjusted analysis). Including gender in this model or analysing separately by gender did not substantially affect the findings.

To initially examine the effect of smoking in early or late pregnancy on adolescent smoking, these two factors were included separately in the logistic model containing the other seven independent predictors or with child aggression at 5 years excluded, as it may be a mediator of this relationship. In the model excluding child aggression at 5 years, smoking in early pregnancy only mildly diminished the effect of maternal smoking at 5 years (adj OR 1.6, 95% CI 1.2, 2.1) and was not itself significant (adj OR 1.2, 95% CI 0.9, 1.6). Including smoking in late pregnancy in the model *(n* = 4275), however, resulted in the effect of smoking at 5 years being reduced further (adj OR 1.4,95% CI 1.0, 1.8), with smoking in late pregnancy being itself a significant predictor of adolescent smoking (adj OR 1.6, 95% CI 1.1, 2.0). To examine the effect

of maternal smoking when the child was 14 years of age, maternal smoking at 14 was then added, together with maternal smoking in early and late pregnancy, to the factors in the logistic model at 5 years predicting adolescent smoking. This analysis was repeated with child aggression at 5 years excluded. The logistic models, both including and excluding child aggression, and containing the seven 5-year predictors together with maternal smoking in early and late pregnancy and at 14 years are shown in Table 3. Smoking at the 14 years follow-up remained a strong predictor (unadj OR 2.8, adj OR 2.6, 95% CI 1.9. 3.5) with smoking in late pregnancy approaching statistical significance, unadj OR 2.1, adj OR 1.5, 95% CI 0.97, 2.2).

A risk score was calculated by summing, in an unweighted manner, the following independent risk factors at 5 years: maternal smoking, maternal alcohol use, non-married, low income, partner ever arrested, four or more children in the household and child aggression. The risk score and its relationship to adolescent smoking are shown in Table 4. There was evidence of a relationship between number of risk factors and risk score, with increasing risks across levels of the risk factor score. If smoking in late pregnancy rather than smoking at 5 years was included in the model, the findings were of similar magnitude to that shown in the table. The risk score was also calculated using the weights from the logistic model. Findings from this were similar to those of the unweighted risk score with positive predictive values being of the same magnitude.

Table 2 Family, maternal and child factors at 5 years related significantly to adolescent smoking.

	Adolescent cigarettes last week						
Factor	*n*	*Nil*	*1-9*	*≥10*	X^2	*df*	*P*
Maternal education*							
High school incomplete	734	85.6	7.2	7.2			
High school complete	2916	88.6	6.5	4.9			
Post high	864	93.3	4.6	2.1	30.5	4	<0.001
Marital status							
Married	3742	90.1	6.0	3.8			
Single	147	85.0	5.4	9.5			
De facto	252	80.6	8.3	11.1			
Other	360	84.4	7.8	7.8	49.0	6	<0.001
Gross family income							
<$16 000 1 year	1034	85.0	7.8	7.2			
$16 000—$25 999	2087	89.2	6.3	4.5			
>$26 000	1290	91.8	5.3	2.9	30.9	4	<0.001
Mother ever arrested							
No	4266	89.5	6.1	4.5			
Yes	138	80.4	8.7	10.9	14.5	2	<0.001
Partner ever arrested							
No	3475	90.2	5.9	3.9			
Yes	708	84.5	7.5	8.1			
No partner	238	87.0	7.1	5.9	27.9	4	<0.001
No. children in household							
<4	3741	89.6	6.1	4.3			

≥4	800	86.5	7.0	6.5	8.1	2	<0.02
Maternal depression							
No	4254	89.2	6.3	4.5			
Yes	273	86.8	5.5	7.7	6.1	2	0.04
Maternal cigarettes/ week							
Nil	2925	91.5	5.4	3.1			
1—9	356	87.1	7.6	5.3			
10—19	553	84.4	8.7	6.9			
≥20	707	83.3	7.2	9.5	73.2	6	<0.001
Maternal alcohol							
Nil	986	91.3	5.1	3.7			
<1 glass/day	3255	88.8	6.4	4.8			
≥1 glass per day	299	83.6	8.7	7.7	14.7	4	0.005
Child externalizing behaviour							
No	4025	89.8	5.9	4.2			
Yes	478	83.3	8.8	7.9	20.5	2	<0.001

*Maternal education level measured at first clinic visit.

DISCUSSION

Adolescent smoking was predicted by a risk score at 5 years involving maternal smoking and alcohol use, non-married status, having a partner who had ever been arrested, having four or more children in the household and child aggression at 5 years. When included with 5- year predictors, smoking in late, although not early, pregnancy was an independent predictor of adolescent smoking and was a stronger predictor than maternal smoking at 5 years. Including information on maternal smoking at the 14 years follow-up indicated that the association between maternal smoking at 5 years and adolescent smoking was related strongly to mothers continuing to smoke at the 14-year follow-up. This would be consistent with adolescent smoking being in part a learned behaviour. The other risk factors at 5 years remained significant in models as predictors of adolescent smoking, although their clinical utility, when combined as a risk score needs to be considered in relation to the reducing numbers of children in higher risk categories. Although inconclusive, there was evidence to suggest that smoking in late pregnancy may exert an independent effect on adolescent smoking separate from maternal smoking at 5 years or at the time of the 14-year follow-up.

Table 3 Predictors of adolescent smoking: measures at 5 years (Model 1) together with model including maternal smoking at other times (Model 2).

5-year predictors	*Unadjusted* OR *(95% CI) n =4257*	*Model 1 * Adjusted OR (95% CI) n =4257*	*Model 2** Adjusted OR (95° CI) n =4188*	*Model 3*** Adjusted OR (95% CI) n =4222*
Maternal cigarette smoking (any)	2.1 (1.7, 2.5)	1.7 (1.4, 2.1)	0.8 (0.6, 1.1)	0.8 (0.6, 1.2)
Maternal alcohol (any)	1.5 (1.2, 2.0)	1.5 (1.1, 1.9)	1.6 (1.2, 2.1)	1.5 (1.1, 2.0)
Non-married	1.9 (1.5, 2.4)	1.4 (1.1, 1.8)	1.4 (1.1, 1.8)	1.4 (1.1, 1.8)
Low income	1.6 (1,3. 2.0)	1.3 (1.0, 1.7)	1.3 (1.0, 1.6)	1.3 (1.0, 1.7)
Partner ever arrested	1.7 (1.3, 2.1)	1.5 (1.2, 1.9)	1.4(1.1, 1.8)	1.5 (1.2, 1.9)
≥4 children in household	1.3 (1.0, 1.6)	1.4(1.1, 1.8)	1.4 (1.1, 1.8)	1.4 (1.1, 1.8)
Child aggression	1.8(1.4,2.4)	1.6(1.2,2.0)	1.7(1.3,2.2)	—

Maternal smoking other times	0.9 (0.6, 1.3)			
Maternal smoking early pregnancy	2.0 (1.6, 2.3)	—	0.8 (0.5, 1.2)	0.9 (0.6, 1.3)
Maternal smoking late pregnancy	2.1 (1.8, 2.5)	—	1.5 (0.97. 2.2)	1.4 (0.9, 2.1)
Maternal smoking at 14 years	2.8 (2.3, 3.3)	—	2.6 (1.9. 3.5)	2.5 (1.9, 3.1)

*Model 1 includes only factors measured at 5 years, **Model 2 includes Model 1 plus maternal smoking early, late pregnancy and when child aged 14 years. ***Model **B:** child aggression 5 years excluded from model.

Table 4 Prediction of adolescent smoking using 5-year risk score (n = 4257).

No. risk factors	*Total*	*Adolescent smokers*		*Criteria*	*Test characteristics*		*PPV*
		n	%		*Sensitivity*	*Specificity*	
Nil	295	14	4.7	Nil			
1	1392	89	6.5	≥1	0.97	0.07	0.11
2	1322	151	11.4	≥2	0.77	0.42	0.14
3	726	95	13.1	≥3	0.45	0.73	0.16
4	387	74	19.1	≥4	0.24	0.89	0.21
5	114	27	23.0	≥5	0.08	0.97	0.27
6	21	10	47.6	≥6	0.02	0.99	0.48

PPV = positive predictive value of test.

Other studies have used similar risk factors in predicting adverse outcomes in children (e.g. Sameroff *et al.* 1987; Fergusson *et al.* 1998; Gutman *et al.* 2003; Rutter 2005) and prenatal exposure to smoking has previously been associated with problems among offspring such as antisocial behaviour (Wakschlag *et al.* 2002), attention deficit hyperactivity disorder (Linnet *et al.* 2003) and child anxiety/depression (Cornelius *et al.* 2000). As a screening instrument for children with the greatest number of risk factors, it has limited utility. For example, for those with six risk factors, the sensitivity and specificity were such that approximately 50% would have taken up smoking but there were only 21 adolescents in this group. For the greater number of children with fewer risk factors, however, the clinical usefulness of the score may lie in providing an early warning sign to identify children who may benefit from early intervention. The benefits of interventions targeting maternal smoking, as well as other risk factors, may therefore extend beyond smoking to a range of other adverse outcomes. If early intervention is not provided for children who exhibit problem behaviours, such children are at high risk of adolescent antisocial behaviour and adult criminality (Fergusson *et al.* 1994; Farrington & Loeber 2000).

The possibility of a direct effect of prenatal smoking on adolescent smoking is consistent with two earlier prospective studies that measured both prenatal and current maternal smoking when offspring were aged 10 (Cornelius *et al.* 2000) and 15—17 years (Kandel *et al.* 1999). In contrast to the current study, however, neither of these included parental and family problems among the range of potential confounding factors examined. The reduction of the OR when smoking was included in the model, and the 95% confidence level approaching, although not excluding the null value suggest chance or unexplained confounding, are also possible explanations for the findings.

The study is not without its limitations, such as potential underreporting of smoking by mothers and adolescents. Reports vary as to the accuracy of self-reported smoking among pregnant women. Buka *et al.* (2003) found significant agreement between self-reported smoking and serum cotinine levels (the major metabolite of nicotine) and earlier studies that have also assessed serum cotinine suggest that adults give accurate self- reports (Pojer *et al.* 1984: Pierce *et al.* 1987). Less favourable results have been reported, however, in a population cohort based sample of pregnant women where half of the women systematically under-reported the amount they smoked (Ford *et al.* 1997). Although biological measures of tobacco use were not used in our study, the information was gathered in circumstances designed to maximize the accuracy of the data (e.g. assurances of confidentiality, the clinical setting, detailed questions, and trained interviewers). Self-reports by young adolescents provide reasonable estimates of actual smoking (Dolcini *eta]*. 2003), and more than 90% agreement between cotinine measurement and adolescent self- reports has been found (Luepker *et al.* 1989).

Loss to follow-up is a further potential weakness. If the risk factors and poor outcomes considered here were less prevalent among those lost to follow up, our results would be biased, over—estimating the association between the identified risk factors and children's tobacco use at age 14. However, as we have found that mothers lost to follow-up in our study are more likely to be younger, single, less educated, socially more disadvantaged and their children of lower birthweight than those in the study group, this seems unlikely. Indeed, it is more likely that those lost to follow-up would exhibit higher rates of tobacco use at age 14, and therefore that the associations presented here would be a conservative estimate (Najman *et al.* 2005). Overall, the fact that we found little difference between the imputed and non-imputed results suggests that attrition is unlikely to have biased our findings substantively in either direction. Finally, it should be noted that the ideal test of an association between maternal and child smoking should be conducted within a genetically informative research design, which was not possible within the current study.
The current findings have a number of strengths in that we have followed adolescents since the time of nicotine exposure *in utero,* thus providing a more complete picture of the predictors of initiation. Further, the statistical analyses fully utilized the longitudinal nature of the data by recording the amount of nicotine and time of exposure during pregnancy and by examining gender differences, as there has been some evidence that the predictors of smoking onset may differ for males and females (Robinson & Klesges 1997). In addition, the study involved a large general population sample and a wide range of variables linked to maternal smoking and child behaviour were taken into account when examining effects. This is particularly noteworthy, given that some previous studies on the effects of maternal smoking were limited by small sample sizes or failure to take into account relevant potentially confounding factors.

In conclusion, adolescent smoking is part of a broad profile of risk factors incorporating maternal smoking during the child's development, maternal alcohol use, non-married status, having a partner who had ever been arrested, having four or more children in the household and child aggression at 5 years. While intervention involving all these factors is difficult for any one of the health professions, the patient—practitioner relationship is one area that could be targeted in order to improve the use of services by pregnant women and mothers of young children, as

well as the success with which smoking is addressed. This is particularly relevant in Australia, where general practitioners check routinely if mothers smoke and seek to give informal advice. The risk factors identified here may provide early warning signs for children who may benefit from early intervention. The possibility of being able to predict other disorders, because of the associations with risk factors related to other adverse outcomes, warrants further investigation.

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